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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.
09/489,220	01/21/00	REIDHAAR-OLSON	J 16528A-03890

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EXAMINER

LU, F

ART UNIT	PAPER NUMBER
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1655

DATE MAILED: 03/30/01

Please find below and/or attached an Office communication concerning this application or proceeding.

Commissioner of Patents and Trademarks

Office Action Summary

Application No.

09/489,220

Applicant(s)

REIDHAAR-OLSON, JOHN F.

Examiner

Frank W Lu

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-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136 (a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).

Status

- 1) ☐ Responsive to communication(s) filed on 05 September 2000.
- 2a) ☒ This action is **FINAL**. 2b) ☐ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 4) ☒ Claim(s) 1-30 is/are pending in the application.
- 4a) Of the above claim(s) 20-27 is/are withdrawn from consideration.
- 5) ☐ Claim(s) _____ is/are allowed.
- 6) ☒ Claim(s) 1-19 and 28-30 is/are rejected.
- 7) ☐ Claim(s) _____ is/are objected to.
- 8) ☐ Claims _____ are subject to restriction and/or election requirement.

Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on _____ is/are objected to by the Examiner.
- 11) ☐ The proposed drawing correction filed on _____ is: a) ☐ approved b) ☐ disapproved.
- 12) ☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. § 119

- 13) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d).
- a) ☐ All b) ☐ Some * c) ☐ None of the CERTIFIED copies of the priority documents have been:
1. ☐ received.
2. ☐ received in Application No. (Series Code / Serial Number) _____.
3. ☐ received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

- 14) ☐ Acknowledgement is made of a claim for domestic priority under 35 U.S.C. & 119(e).

Attachment(s)

- 15) ☒ Notice of References Cited (PTO-892)
- 16) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
- 17) ☐ Information Disclosure Statement(s) (PTO-1449) Paper No(s) _____.
- 18) ☐ Interview Summary (PTO-413) Paper No(s). _____.
- 19) ☒ Notice of Informal Patent Application (PTO-152)
- 20) ☐ Other: _____.

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DETAILED ACTION

Response to Amendment

1. Applicant's response to the office action filed on January 8, 2001 has been entered as Paper No: 8. The claims pending in this application are claims 1-30 with nonelected claims 20-27. Rejection and or objection not reiterated from the previous office action are hereby withdrawn. The following rejections are based on amendment.

Election/Restriction

2. This application contains claims 20-27 drawn to an invention nonelected with traverse in Paper No. 4. A complete reply to the final rejection must include cancellation of nonelected claims or other appropriate action (37 CFR 1.144) See MPEP § 821.01.

Drawings

3. Applicant's request that "applicant will provide formal drawings upon notification of allowable subject matter" has been granted by the examiner.

Information Disclosure Statement

4. In page 14, third paragraph of applicant's remarks, applicant stated that "the office action did not have the Information Disclosure Statement (Form 1449) submitted on June 6, 2000, attached to it". However, the examiner noticed there was no Information Disclosure Statement

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(Form 1449) inside the file and there was no record to show that Information Disclosure Statement (Form 1449) submitted on June 6, 2000 had been entered into PALM system.

Claim Objections

5. Claims 7 and 9 are objected to under 37 CFR 1.75(c), as being of improper dependent form for failing to further limit the subject matter of a previous claim. Applicant is required to cancel the claim(s), or amend the claim(s) to place the claim(s) in proper dependent form, or rewrite the claim(s) in independent form.

Note that claim 7 contains F₁F₀-ATPase synthase and NADH dehydrogenase subunit 2 while claim 9 contains Glutathione-S-transferase, heat shock protein 90, cAMP-dependent transcription factor ATF-4 and EST(AI148382) which are not found in claim 1. For the examination purpose, the examiner considered that claim 1 contains all these proteins.

Claim Rejections - 35 U.S.C. § 112

6. The following is a quotation of the second paragraph of 35 U.S.C. 112:

The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

7. Claims 1-19 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention. Note that claims 2-19 are dependent on claim 1.

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Claim 1 is rejected as vague and indefinite because it is unclear what it intended. For example, how a method for detecting a toxic response does not have a step of incubating a test with a toxic material?

8. Claim 1 is rejected under 35 U.S.C. 112, second paragraph, as being incomplete for omitting essential steps, such omission amounting to a gap between the steps. See MPEP § 2172.01. The omitted step is to incubate a test sample with a toxic material.

9. Claim 7 recites the limitation " F_1F_0 -ATPase synthase and NADH dehydrogenase subunit 2" in the claim. There is insufficient antecedent basis for this limitation in the claim.

10. Claim 9 recites the limitation "Glutathione-S-transferase, heat shock protein 90, cAMP-dependent transcription factor ATF-4 and EST(AI148382) " in the claim. There is insufficient antecedent basis for this limitation in the claim.

Claim Rejections - 35 U.S.C. § 102

11. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless --

(b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

12. Claims 1, 7, 11, 14, 18, and 19 are rejected under 35 U.S.C. 102(b) as being anticipated by Luciakova *et al.*, (Eur. J. Biochem. 207, 253-257, 1992).

Luciakova *et al.*, teach the response of several RNA for mitochondrial proteins to growth activation by serum in NIH 3T3 cells. Figures 2 and 3 showed different gene expressions using

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RNA isolated from NIH 3T3 deprivation of serum for 24 hours and time course of serum addition. The steady-state levels of the transcript for one nuclear-encoded respiratory-chain component, F₁-ATPase beta-subunit (water-soluble portion of F₁F₀-ATPase synthase) decreased significantly in quiescent cells and was rapidly restored with similar kinetics after addition of serum while the transcript for one additional nuclear-encoded mitochondrial gene, cytochrome c1 did not respond to serum deprivation or growth activation. These results implied that mitochondrial biogenesis was at least partially regulated through growth-dependent mechanisms (see page 267, abstract and page 255). Serum deprivation or growth activation could be considered as a toxic material.

Therefore, Luciakova *et al.*, teach all limitations recited by claims 1, 7, 11, 14, 18, and 19.

13. Claims 1, 9, 11, 14, 18, and 19 are rejected under 35 U.S.C. 102(b) as being anticipated by Nemoto *et al.*, (Eur. J. Biochem. 233, 1-8, 1995).

Nemoto *et al.*, teach to make Glutathione-S-transferase (GST) and GST-HSP 90 α (GST-heat shock protein 90) fusion protein. GST and GST-HSP 90 α were in lanes 1 and 7 of Figure 1 (page 2). This has been well known that GST and GST-fusion protein were made by IPTG induction (see Nemoto *et al.*, J. Steroid Biochem. Molec. Biol. 50, 225-233, 1994, especially see page 226, right column, fourth paragraph). Although Nemoto *et al.*, did not show that the expressions of GST and GST-HSP 90 α in control sample as described claim 1, in the absence of convincing evidence to the contrary the claimed invention, these limitations is considered as

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inherent to the reference taught by Nemoto *et al.*, since no GST protein was expression in the absence of IPTG. IPTG could be considered as a toxic material and the expression levels of nucleic acids could be considered either in transcriptional or translational level.

Therefore, Nemoto *et al.*, teach all limitations recited by claims 1, 9, 11, 14, 18, and 19.

Claim Rejections - 35 U.S.C. § 103

14. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negated by the manner in which the invention was made.

15. Claims 1, 5-11, 14, 15, 18, and 19 are rejected under 35 U.S.C. 103(a) as being unpatentable over Diel *et al.*, (J. Steroid Biochem. Molec. Biol. 55, 363-373, 1995) in view of and Fagan *et al.*, (J. Biol. Chem. 264, 20513-20517, 1989).

Diel *et al.*, teach identification of estrogen regulated genes in Fe33 rat hepatoma cells by differential display polymerase chain reaction. Three genes of known sequences including insulin-like growth factor binding protein-1 (IGFBP-1) were detected by the ddRT-PCR approach. Effects of ethinyl estradiol on the mRNA levels of these genes were confirmed by "Northern-blot" analysis. If given in combination with dexamethasone and glucagon, ethinyl estradiol caused 40-fold increases in the mRNA steady state level of IGFBP-1 in Fe33 cells 24 h after addition of hormone (see abstract in page 363 and Figures 1, 3, and 4 in pages 365, 367, and 368).

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Fagan *et al.*, teach regulation of ornithine aminotransferase (OAT) in retinoblastomas. They found that two retinoblastoma strains, Y79 and RB355, had approximately 5-fold increases in OAT protein and mRNA levels. Note that, although OAT is expressed in most tissues of the rat, liver, kidney, and retina had the highest levels of OAT activity (see abstract in page 20513, Figures 1 and 2 in pages 20514 and 20515).

Diel *et al.*, and Fagan *et al.*, do not disclose to analyze two different genes, ie. insulin-like growth factor binding protein-1 and ornithine aminotransferase in a single assay.

Therefore, in the absence of an unexpected result, it would have been *prima facie* obvious to one having ordinary skill in the art at the time the invention was made to have examined estrogen effects in the regulation of gene expression of two different genes such as IGFBP-1 and OAT in test cells originated from liver as suggested by Diel *et al.*, and Fagan *et al.*. One having ordinary skill in the art would have motivated to modify and combine above method together in order to identify new estrogen regulated gene from a liver originated cell because least two different genes in claims 1 and 5-10 could be found in liver and have been well known in the art. For example, enzymes for glucose and lipid metabolism (ie. pyruvate dehydrogenase), proteins for oxidation phosphorylation (ie. cytochrome c1) (see any Biochemistry textbook), and defender against cell death 1 (see Hong et al., Mol. Cell. Biol. 17, 2151-2157, 1997, especially Figure 5). One having ordinary skill in the art at the time the invention was made would have been a reasonable expectation of success to combine these methods together because all of these methods are known in the art and are easy to use.

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16. Claims 2-4 are rejected under 35 U.S.C. 103(a) as being unpatentable over Diel *et al.*, (1995) and Fagan *et al.*, (1989) as applied to claims 1, 5-11, 14, 15, 18, and 19 above, and further in view of Li *et al.*, (Cell Biol. Int. Rep. 13, 619-624, 1989).

The teachings of Diel *et al.*, and Fagan *et al.*, have been summarized previously, *supra*.

Li *et al.*, teach estrogen-induced expression of mouse lactate dehydrogenase-A gene in uterus of immature mice. Note that lactate dehydrogenase also expresses in liver (see Textbook of Biochemistry with clinical correlations, edited by Thomas M. Devlin, Third Edition, page 296, 1992).

Diel *et al.*, Fagan *et al.*, and Li *et al.*, do not disclose to analyze three different genes, ie. insulin-like growth factor binding protein-1, ornithine aminotransferase, and lactate dehydrogenase-A in a single assay.

Therefore, in the absence of an unexpected result, it would have been *prima facie* obvious to one having ordinary skill in the art at the time the invention was made to have examined estrogen effects in the regulation of gene expression of three or five or at least ten different genes in test cells originated from liver as suggested by Diel *et al.*, Fagan *et al.*, and Li *et al.*. One having ordinary skill in the art would have motivated to modify and combine above method together in order to identify new estrogen regulated gene from a liver originated cell such as Fe33 cells because at lest ten different genes in claim 1 could be found in liver . For example, enzymes for glucose and lipid metabolism (ie. pyruvate dehydrogenase) and proteins for oxidation phosphorylation (ie.cytochrome c1) (see any Biochemistry textbook). One having ordinary skill in the art at the time the invention was made would have been a reasonable expectation of success

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to combine these methods together because all of these methods are known in the art and are easy to use.

17. Claims 12 and 13 are rejected under 35 U.S.C. 103(a) as being unpatentable over Diel *et al.*, (1995) and Fagan *et al.*, (1989) as applied to claims 1, 5-11, 14, 15, 18, and 19 above, and further in view of Desjardins *et al.*, (Cancer Lett., 131, 201-207, 1998).

The teachings of Diel *et al.*, and Fagan *et al.*, have been summarized previously, *supra*.

Diel *et al.*, and Fagan *et al.*, do not disclose to use HepG2 cells.

Desjardins *et al.*, do teach to study the expression of different genes in the presence of a toxic material using HepG2 cells (see page 201, abstract). Note that HepG2 cells are human hepatoma cells (see page 201, left column).

Therefore, in the absence of an unexpected result, it would have been *prima facie* obvious to one having ordinary skill in the art at the time the invention was made to have examined estrogen effects in the regulation of gene expression of two different genes such as IGFBP-1 and OAT as suggested by Diel *et al.*, and Fagan *et al.* in HepG2 cells as suggested by Desjardins *et al.*, One having ordinary skill in the art would have motivated to modify and combine above method together because the simple replacement of one kind of liver cell from another kind of liver cell (i.e. HepG2 cell) would have been, in the absence of an unexpected result, *prima facie* obvious to one having ordinary skill in the art at the time the invention was made. Furthermore, the motivation to make the substitution cited above, arises from the expectation that the prior art elements will perform their expected functions to achieve their expected results when combined

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for their common known purpose. Support for making the obviousness rejection comes from the M.P.E.P. at 2144.07 and 2144.09.

Also note that there is no invention involved in combining old elements in such a manner that these elements perform in combination the same function as set forth in the prior art without giving unobvious or unexpected results. *In re Rose* 220 F.2d. 459, 105 USPQ 237 (CCPA 1955).

18. Claim 16 is rejected under 35 U.S.C. 103(a) as being unpatentable over Diel *et al.*, (1995) and Fagan *et al.*, (1989) as applied to claims 1, 5-11, 14, 15, 18, and 19 above, and further in view of Schena *et al.*, (Proc. Natl. Acad. Sci. USA, 93, 10614-10619, 1996).

The teachings of Diel *et al.*, and Fagan *et al.*, have been summarized previously, *supra*.

Diel *et al.*, and Fagan *et al.*, do not disclose to use a cDNA array.

Schena *et al.*, teach parallel human genome analysis using microarray-based expression. In this study, a total of 1056 cDNA, representing 1046 human clones and 10 *arabidopsis* control, were arrayed in 1.0-cm² areas (page 10614, right column, second paragraph). Microassays were used to examine the cellular effects of phenol ester treatment in cultured human T (Jurkat) cells. Untreated and phenol ester-treated cells were harvested and lysed, and total mRNA from the two cell samples was labeled by reverse transcriptase incorporation of fluorescein- and cy5-dCTP, respectively. In a second set of labeling reactions, the fluorescent groups were "swapped" such that sample from control and phenol ester-treated samples were labeled with cy5- and fluorescein-dCTP, respectively (page 10616, Figure 2B). Each pair of fluorescent probes was hybridized to a 1056-element microarray. A total of six array elements displayed ≥ 2.0 fold elevated signals with

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probes from phorbol ester-treated cells relative to control samples (page 10617, left column, second paragraph and Figure 2B in page 10616). The identity of the phenol ester-induced genes were confirmed by DNA sequencing (page 10617, left column, third paragraph).

Therefore, in the absence of an unexpected result, it would have been *prima facie* obvious to one having ordinary skill in the art at the time the invention was made to have examined estrogen effects in the regulation of gene expression of two or more different genes such as IGFBP-1 and OAT as suggested by Diel *et al.*, and Fagan *et al.*, in a cDNA array as suggested by Schena *et al.*. One having ordinary skill in the art would have motivated to modify and combine above method together because: (1) the method to make a cDNA array and genes listing in claim 1 have been well known in the art at the time the invention was made; and (2) the simple replacement of one well known detection method from another well known detection method (i.e. cDNA array) would have been, in the absence of an unexpected result, *prima facie* obvious to one having ordinary skill in the art at the time the invention was made. Furthermore, the motivation to make the substitution cited above, arises from the expectation that the prior art elements will perform their expected functions to achieve their expected results when combined for their common known purpose. Support for making the obviousness rejection comes from the M.P.E.P. at 2144.07 and 2144.09.

Also note that there is no invention involved in combining old elements in such a manner that these elements perform in combination the same function as set forth in the prior art without giving unobvious or unexpected results. *In re Rose* 220 F.2d. 459, 105 USPQ 237 (CCPA 1955).

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19. Claim 17 is rejected under 35 U.S.C. 103(a) as being unpatentable over Diel *et al.*, (1995) and Fagan *et al.*, (1989) as applied to claims 1, 5-11, 14, 15, 18, and 19 above, and further in view of Zamorano *et al.*, (Neuroendocrinology 63, 397-407, May 1996).

The teachings of Diel *et al.*, and Fagan *et al.*, have been summarized previously, *supra*.

Diel *et al.*, and Fagan *et al.*, do not disclose to quantitative RT-PCR.

Zamorano *et al.*, reviewed quantitative RT-PCR. One of advantage of this technique is to measure mRNA levels in small amounts of tissue or even in single cells (page 406, left column, second paragraph).

Therefore, in the absence of an unexpected result, it would have been *prima facie* obvious to one having ordinary skill in the art at the time the invention was made to have examined estrogen effects in the regulation of gene expression of two or more different genes such as IGFBP-1 and OAT as suggested by Diel *et al.*, and Fagan *et al.*, using quantitative RT-PCR as suggested by Zamorano *et al.*. One having ordinary skill in the art would have motivated to modify and combine above method together because the simple replacement of one well known detection method from another well known detection method (i.e. quantitative RT-PCR, see Zamorano *et al.*, for the cited references) would have been, in the absence of an unexpected result, *prima facie* obvious to one having ordinary skill in the art at the time the invention was made. Furthermore, the motivation to make the substitution cited above, arises from the expectation that the prior art elements will perform their expected functions to achieve their expected results when combined for their common known purpose. Support for making the obviousness rejection comes from the M.P.E.P. at 2144.07 and 2144.09.

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Also note that there is no invention involved in combining old elements in such a manner that these elements perform in combination the same function as set forth in the prior art without giving unobvious or unexpected results. *In re Rose* 220 F.2d. 459, 105 USPQ 237 (CCPA 1955).

20. Claim 28-30 are rejected under 35 U.S.C. 103(a) as being unpatentable over Diel *et al.*, (1995), Fagan *et al.*, (1989) and Li *et al.*, (1989) as applied to claims 1-11, 14, 15, 18, and 19 above, and further in view of Martin *et al.*, (BioTechnique 21, 520-524, September 1996).

The teachings of Diel *et al.*, Fagan *et al.*, and Li *et al.*, have been summarized previously, *supra*. Note that Li *et al.*, teach to examine estrogen-induced expression of mouse lactate dehydrogenase A gene using mouse lactate dehydrogenase A promoter and cat fusion gene (see forth paragraph of page 620 and first paragraph in page 621).

Diel *et al.*, Fagan *et al.*, and Li *et al.*, do not disclose the determination of gene expression using different reporter systems.

Martin *et al.*, do teach the determination of gene expression using different reporter systems (ie. luciferase and beta-galactosidase). Note that both independent and combined (Dual-Light) detection methods for cells cotransfected with luciferase and beta-gal reporter genes are sensitive enough to determine gene expression (see abstract in page 520 and Table 1 in page 522).

Therefore, in the absence of an unexpected result, it would have been *prima facie* obvious to one having ordinary skill in the art at the time the invention was made to have examined estrogen effects in the regulation of gene expression of three or more different genes such as

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IGFBP-1, OAT, lactate dehydrogenase A using promoters from different genes as suggested by Diel *et al.*, Fagan *et al.*, and Li *et al.*, and using different reporter systems as suggested by Martin *et al.*. One having ordinary skill in the art would have motivated to modify and combine above method together because: (1) promoters of at least three of genes listed in claims 28-30 have been well known in the art, i.e. OAT and IGFBP-1 (see Biochim. Biophys. Acta, 1132, 214-218, 1992 and Endocrinology 134, 736-743, 1994); and (2) the simple replacement of one well known detection method from another well known detection method (i.e. using reporter gene) would have been, in the absence of an unexpected result, *prima facie* obvious to one having ordinary skill in the art at the time the invention was made. Furthermore, the motivation to make the substitution cited above, arises from the expectation that the prior art elements will perform their expected functions to achieve their expected results when combined for their common known purpose. Support for making the obviousness rejection comes from the M.P.E.P. at 2144.07 and 2144.09.

Also note that there is no invention involved in combining old elements in such a manner that these elements perform in combination the same function as set forth in the prior art without giving unobvious or unexpected results. *In re Rose* 220 F.2d. 459, 105 USPQ 237 (CCPA 1955).

Conclusion

21. Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

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A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

22. No claim is allowed.

23. Papers related to this application may be submitted to Group 1600 by facsimile transmission. Papers should be faxed to Group 1600 via the PTO Fax Center located in Crystal Mall 1. The faxing of such papers must conform with the notices published in the Official Gazette, 1096 OG 30 (November 15, 1988), 1156 OG 61 (November 16, 1993), and 1157 OG 94 (December 28, 1993)(See 37 CAR § 1.6(d)). The CM Fax Center number is either (703) 308-4242 or (703)305-3014.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Frank Lu, Ph.D., whose telephone number is (703) 305-1270. The examiner can normally be reached on Monday-Friday from 9 A.M. to 5 P.M.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, W. Gary Jones, can be reached on (703) 308-1152.

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Any inquiry of a general nature or relating to the status of this application should be directed to the Chemical Matrix receptionist whose telephone number is (703) 308-0196.

Frank Lu
March 26, 2001

A handwritten signature in black ink, appearing to read 'EWhisenant', with a stylized flourish at the end.

Ethan Whisenant, Ph.D.
Primary Examiner (FSA)